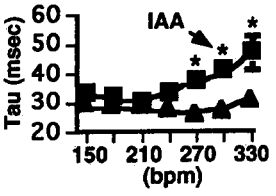
paced, isovolumically-beating pig hearts (N = 13, < 3 days of age), underwent non-recirculating, retrograde-aortic perfusion (37ºC) with a well-oxygenated buffer solution containing 2% BSA, insulin (100 μU/ml) and either: 5.5 mM glucose (Control) or 5.5 mM glucose, 50 μM iodoacetate to inhibit glycolysis and 5.5 mM pyruvate to sustain oxidative metabolism (IAA). Left ventricular (LV) balloon volume was fixed at ≅ 0.07 ml/g_{wet}. LV peak systolic pressure (PSP), end diastolic pressure (EDP), $-dP/dt_{max}$ and the relaxation time constant (Tau) were determined. Hearts were perfused with a pressure of 60 mmHg during 3 consecutive periods: 1) Baseline, HR 150 bpm (30 min.), 2) Generation of HR-response curve and 3) Tachycardia (Tach), HR 300 bpm (30 min.). During Baseline, PSP, EDP, $-dP/dt_{max}$ and Tau averaged 122 ± 6 mmHg, 5.7 ± 0.3 mmHg, 1279 ± 102 mmHg/sec and 30 ± 3 msec; and 127 ± 8 mmHg, 6.7 ± 0.7 mmHg, 1233 ± 127 mmHg/sec and 32 ± 2 msec, for Control and IAA, respectively. During Tach, the values were 84 ± 4 mmHg, 9.7 ± 1.7 mmHg, 1000 ± 92 mmHg/sec and 29 ± 5 msec; and 73 ± 4 mmHg, 31.8 ± 4.1* mmHg, 546 ± 89* mmHg/sec and 68 ± 6* msec, for Control and IAA, respectively. For IAA, the HR-response curve was markedly shifted (Fig).



For each HR studied, PSP *did not* significantly differ between Control and IAA. In conclusion, inhibition of glycolysis in neonatal pig hearts severely impairs LV relaxation.

1043-65 Sarcoplasmic Reticulum Calcium-ATPase mRNA Level Decreases in Pressure-Overloaded Fetal Lamb Ventricle

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The mRNA concentration of the Sarco(endo)plasmic Reticulum Calcium-ATPase (SERCA-2a) increases in the left ventricle (LV) during ontogenic development but decreases during pressure overload-induced hypertrophy. Because an increase in LV afterload is common in congenital heart diseases, we investigated whether such an increase alters the normal ontogenic program of SERCA-2a gene expression in the fetal lamb LV. In 9 fetal lambs, we performed a preductal coarctation of the aorta by banding the aortic arch (CoA) *in utero* at mid-gestation and a sham-operation in 9 twins (T). All fetuses were studied 4 weeks later for LV pressure, anatomical, histological and molecular measurements. LV SERCA-2a mRNA concentration was assessed by Northern blot hybridization using a rat cDNA probe and normalized to 18S ribosomal RNA. Surgery resulted in a severe coarctation of the aorta in all CoA but not in T as assessed by a marked decrease in the diameter of the preductal aorta (0.8 ± 0.4 mm vs 4.2 ± 0.6 mm, p < 0.0001) associated with a major increase in the pressure gradient through the aortic arch (31.9 ± 6.5 mmHg vs 2.4 ± 1.9 mmHg; p < 0.0001) in the former compared to the latter. This was associated with a 65% increase in the LV weight to body weight ratio in CoA as compared with T (p < 0.001). The two groups did not differ in their mean LV pressures, peak positive and negative dP/dt and LV cardiomyocyte transverse diameter. Northern blot analysis using the SERCA-2a specific cDNA revealed a single sharp hybridization band around 4.4 Kb for each fetal RNA sample. Importantly, the LV concentration of SERCA-2a mRNA in CoA decreased to 28.6% its value in T (p = 0.003). *Conclusion:* Regarding SERCA-2a gene expression in the lamb LV, the pathological process of pressure overload-induced hypertrophy overrides and/or slows down the physiological process of the ontogenic maturation without altering cardiac pump function

1043-66 Impaired Pulmonary Vascular Relaxation in Tetralogy of Fallot

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Pulmonary endothelial function and smooth muscular response to nitoric oxide donor agents in Tetralogy of Fallot (TF) remains unknown, although the abnormalities are well documented in patients with pulmonary hypertension. Accordingly, we examined endothelial dependent and non-dependent vasodilative ability of pulmonary artery in 7 patients (1.8 ± 0.9 years) with TF and 6 controls with normal pulmonary hemodynamics. Flow velocity (FV) and integral (FI) were measured with use of a Doppler-tip flow wire (Cardiometrics) placed in peripheral pulmonary artery during incremental infusion of acetylcholine (ACH 10^{-8}, 10^{-7}, 10^{-6}M) and nitroglycerine (0.5, 1.0 μg/kg/min) into the artery. In contrast to the dose dependent increase in pulmonary flow indices (FV and FI) to ACH and NTG stimulation, there is less increase in FV and FI in TF patients. Maximum increase ratio of both indices (% FV and % FI) to each stimulation were significantly depressed in TF patients than in control (ACH:100 ± 21% vs 153 ± 20% in % FV, 99 ± 18% vs 146 ± 28% in % FI) (NTG: 121 ± 14% vs 155 ± 23% in % FV, 119 ± 20% vs 138 ± 18% in % FI). In addition, % FV and % FI to ACH were significantly correlated with pulmonary to systemic flow ratio (Qp/Qs) (r = 0.84 in % FV, r = 0.79 in % FI) and mixed venous saturation (r = 0.71 in % FV, r = 0.66 in % FI), and % FV to NTG was also correlated with Qp/Qs (r = 0.68) and mixed venous saturation (r = 0.50). These data suggest that decreased and hypoxic blood flow in TF patients attenuates both endothelial dependent and non-dependent pulmonary vascular relaxation.

1043-67 Endogenous Nitric Oxide-Related Compounds in Whole Blood are Increased in Patients with Congenital Heart Defects with Pulmonary Hypertension

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Inhalation of nitric oxide (NO) has been widely used for acute post operated patients with congenital heart defects (CHD) with pulmonary hypertension (PH). Large left to right shunt with PH in CHD results in imbalance between vasoconstricting factors and NO endogenously produced by pulmonary artery endothelial cells. We determined NO-related compounds in whole blood of CHD patients and evaluated the relationship between PH and endogenous NO. Twenty three patients (age 6 months to 5 years) were divided into three groups, [group 1]: no shunt, PH (−), [group 2]: shunt (+), PH (−), Pp/Ps < 0.4, [group 3]: shunt (+), PH (+), Pp/Ps > 0.4. Whole blood were taken from right atrium, pulmonary artery, wedge pulmonary artery and aorta at cardiac catheterization, then blood samples were stored at −20ºC until assay. All NO-related compounds in blood were determined by thermal liberation of NO from nitrosyl complexes with denaturing proteins and finally detection of resulting nitrate ion by chemiluminescence after an enzymatic conversion to nitrite using Sivers NO analyzer. Endogenous nitric oxide-related compounds in whole blood are significantly increased in group 3 patients (84.2 ± 9.3 μM) compared with group 1 and 2 (21.1 ± 1.9μM, 28.9 ± 6.4 μM, respectively) (P < 0.01). These results suggest that high flow left to right shunts might be leading to elevation of endogenous NO production by pulmonary artery endothelial cells.

1043-68 Predictive Value of Serum Cardiac Troponin T (cTnT) in Pediatric Patients at Risk for Myocardial Injury

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Biochemical markers of myocardial damage are not routinely used in children at risk for myocardial injury. We investigated the clinical utility of cTnT levels (CARDIAC T, Boehringer Mannheim Corp.) in pts at a pediatric hospital (0.1–30 yrs old) undergoing cardiovascular surgery (n = 19) or receiving doxorubicin for acute lymphoblastic leukemia (ALL) (n = 11) where detectable cTnT elevations were ≥ 0.03 ng/ml. In the 19 surgical pts, a significant correlation was noted between the surgical severity score determined prior to measuring cTnT (increasing severity: vascular, atrial, valvular, ventricular, ventricular resection) and the level of post-operative cTnT 0.3, 1.1, 0.7, 4.1, ≥ 11.2 ng/ml respectively) (r = 0.79, p < 0.0001). The post-operative cTnT level was significantly (p = 0.0083) associated with children who completed cardiovascular surgery with an open chest (n = 2, mean cTnT ≥ 17.7 ng/ml) compared with those with a closed chest (n = 16, mean cTnT = 1.73 ng/ml). The pre-operative cTnT level significantly (p = 0.007) predicted post-op survival; 15 pts had undetectable cTnT, 1 pt had a cTnT < 0.1 ng/ml, and cTnT was ≥ 0.1 ng/ml in the only 2 pts who died. Similarly, in the 11 ALL pts, cTnT elevation following initial doxorubicin predicted LV dilation and wall thinning by echocardiography 9 months (mean) later. The LV dimension z score adjusted for BSA was −0.72 SD for the 4 pts with cTnT < 0.03 ng/ml and 1.08 SD for the 7 pts with cTnT ≥ 0.03 ng/ml (r = 0.80 when the variable was treated as continuous, p = 0.003). LVED posterior wall thickness z score adjusted for BSA was −0.13 SD in the low cTnT group and −0.66 SD in the high cTnT group (r = 0.61, p = 0.044). In conclusion, elevations of serum cTnT in children appear to quantitatively relate to the severity of myocardial damage and predict subsequent subclinical and clinical morbidity and mortality. cTnT

may be useful for monitoring and determining interventions for children at risk of myocardial damage.

1043-69 A Mathematical Method of Predicting Peak to Peak Gradients From Doppler Echocardiography in Obstructive Lesions

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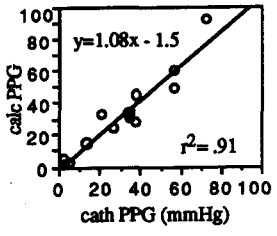
Background: We report a computer-assisted method of converting doppler peak instantaneous gradients (dPIG) to peak to peak gradients (calc PPG) that are reliably close to those measured at catheterization (cath PPG).

Methods and Results: In 16 patients (ages 5 months to 12 years) with various left-sided obstructive lesions (6 coarctations, 7 aortic stenoses, 3 subaortic stenoses), we obtained doppler measurements of blood flow velocity across the obstruction simultaneously with brachial or femoral plesmographic waveforms and cuff pressures at the time of cardiac catheterization. The cath PPGs ranged from 2 to 72 mmHg. While blinded to the results of the catheterization, we applied our computer program with curve-fitting capabilities to the doppler and plesmographic images to develop equations describing the dPIG and the pre- or post-obstruction arterial waveform as functions of time. These equations were substituted into the Bernoulli equation producing a solution describing the unknown pressure waveform. The calculated PPG was obtained by solving or the maximum of the equation for this waveform.

This method consistently provided the closest approximation of the cath PPG compared to the standard echocardiographic doppler measurements.



Analysis of Regression with Cath PPG

Condition and Type of	Data	Slope	Intercept	r^2
Unsedated Echo	dPIG	1.3	19	0.39
	dMG	0.8	8	0.37
Sedated Echo	dPIG	1.3	7	0.82
	dMG	0.9	−4	0.78
This Method	calc PPG	1.08	−1.5	0.91

dPIG: doppler peak instantaneous gradient. dMG: doppler mean gradient

Conclusion: This method is an easy and reliable non-invasive method of predicting the severity of certain obstructive lesions.

1044 The 12-Lead ECG in Acute Myocardial Infarction

Wednesday, March 19, 1997, 9:00 a.m.–11:00 a.m.
Anaheim Convention Center, Hall E
Presentation Hour: 9:00 a.m.–10:00 a.m.

1044-103 ECG Criteria Differentiating Between Proximal Versus Distal Occlusions of the Left Anterior Descending Coronary Artery

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Proximal left anterior descending (LAD) coronary artery disease has a poor prognosis, especially in LAD obstruction proximal of the first septal perforator (S1). To assess the value of the ECG as a non-invasive predictor of occlusion site, 100 patients (pts) with a first anterior myocardial infarction minimally having ST elevation (ST↑) of ≥ 2 mm in V2 and V3 were included in the study. Changes in the ST segment and the development of right bundle branch block (RBBB) were evaluated and correlated with the exact LAD occlusion site as determined by coronary angiography.

Results: 46 pts had an occlusion of the LAD proximally of S1 and 54 pts distally of S1. The presence nor the amount of ST↑ in the leads V1–V4 correlated with the site of occlusion. ST↑ in aVR, ST depression (ST↓) in V5 and the development of RBBB were highly specific (spec) for proximal LAD occlusion, although sensitivity (sens) of the latter two criteria was low. Absence of ST↓ in the inferior leads, particularly in aVF, was associated with distal LAD occlusion.

	Sens	Spec	PA+	PA−
$ST\uparrow_{V1}$	0.89	0.16	0.48	0.64
$ST\uparrow_{aVR}$	0.35	0.96	0.89	0.63
RBBB	0.15	1.00	1.00	0.58
$ST\uparrow_{aVR}$ and/or RBBB	0.46	0.96	0.91	0.68
$ST\downarrow_{V5}$	0.15	0.98	0.88	0.58
absence of $ST\downarrow_{aVF}$	0.41	0.91	0.85	0.57

(PA+ and PA−: positive and negative predictive accuracy)

Conclusions: In anterior myocardial infarction, presence of ST↑ in aVR, ST↓ in V5 and RBBB strongly predicts proximal LAD occlusion while absence of ST↓ in aVF strongly predicts distal occlusion.

1044-104 Noninvasive Identification of the Infarct Related Artery From Zone Activity in the True Peak Surface 12 Lead ECG

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Determination of the infarct related artery (IRA) in acute MI has prognostic value for therapeutic agents including thrombolytics. Continuous 12-lead ECG recordings from 437 patients with angiographically identified IRAs from the AMI TAMI-9 (n = 189), GUSTO-1 (n = 139), DUCCS-2 (n = 25), and IMPACT-I (n = 84) trials were reviewed to determine true peak ST deviation and model surface ECG zone activity patterns vs. IRA. All trials required ST deviation to enroll. Blinded core lab read ST activity as elevation, depression, or none (J + 60 > 100 uV deviation from peak ECG vs. baseline) in precordial zones defined as ANT = V2–4; INF = II, III, aVF; HLAT = I, aVL; LLAT = V5, V6. Angiogram data revealed 175 LAD, 208 RCA & 54 CIRC IRAs. In these pts, 40/81 possible zone activity patterns (3 activity patterns × 4 precordial zones) were observed. All patterns with ANT elevation (21/40) with any other activity were 92% sensitive, and 66% specific for IRA = LAD. Chi Square analysis of IRA = Non-LAD pts, LLAT ($X2 = 10.06$, $p = 0.007$) and HLAT ($X2 = 7.55$, $p = 0.023$) zone activity yielded some separation of RCA from CIRC, generally in the presence of INF ST elevation. Considerable overlap was notable between these groups. Thus, single zone activity on standard 12 lead ECG at true peak ST deviation identifies LAD IRA, but separates RCA from CIRC poorly. Accuracy improves with the use of additional ECG zone activity over the precordium.

1044-105 ST II/III: A New Electrocardiographic Criteria for the Identification of the Culprit Vessel in Inferior Wall Myocardial Infarction

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The identification of the culprit vessel by ECG may be difficult or even impossible in inferior wall MI (IWMI). The aim of this study was to determine if the ratio of magnitude of ST elevation in leads II and III (ST II/III) would differentiate between RCA and LCX as the culprit vessel. *Methods:* We retrospectively identified all patients with IWMI who underwent coronary angiography over a period of 2.5 years. The ST II/III ratio was determined from the presenting ECG. A ratio < 1 (ST elevation in lead III > II) indicated the RCA as the culprit vessel and a ratio > 1 (ST elevation in lead II > III) the LCX. The culprit vessel was identified from the coronary angiogram by an experienced angiographer who was blinded to the ECG findings. Patients were excluded if there was significant disease in both arteries. *Results:* A total of 106 patients were identified. The table shows the coronary involvement and the ST II/III ratio.

	RCA	LCX
ST II/III < 1	83	4
ST II/III > 1	0	19

The ST II/III ratio correctly differentiated between RCA and LCX as the culprit vessel in 96% of patients. *Conclusion:* The ST II/III ratio is a highly accurate new criteria to differentiate the culprit vessel in IWMI.